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HYDATID DISEASE.

M. D. Thesis. (G. R. Burnett.)



HYDATID DISEASE.

Advances of recent years have greatly helped towards our understanding of Hydatid disease; there are, however, still some aspects of the condition that require further study, before we can hope for a reduction of the mortality, or still better, the total eradication of the disease.

Hydatid disease is comparatively rare in this country, but in certain parts of the world it is responsible for many deaths and much disability.

A series of cases occurring in the sheep farming area of the English Lake district, impress on one, the necessity for a more serious consideration of the disease and for a more thorough preventive measure.

Before entering into this aspect of the condition however, it would be well to review our present knowledge of the history, prevalence, and cause of the disease.

Historical.

The frequent references to Hydatid disease in Medical Literature throughout all ages, prove that this is no new condition. Hippocrates (1) recognised it as a Pathological condition and writes:- "When the liver is filled with water and bursts into the epiploon, the belly is filled with water and the patient dies."

Aretaeus, who lived about the middle of the second century, refers to the disease as a form of dropsy, due to small and numerous bladders full of fluid.

In 1684, the animal nature of the bladder worms was first described by Redi, and in 1685 and 1691 respectively, Hartmann and Tyson confirmed Redi's hypothesis. A hundred years later, Pallas recognised that Hydatid disease was due to a specific form of Taenia.

The feeding experiments of Küchenmeister in 1851, identified the bladder worms, as the larval stage of certain tape worms.

The Parasite.

- (1.) The *Taenia Echinococcus*, measuring 3-5 m.m. in length and 2-3 m.m. in breadth, is the smallest tape worm known.
- (2.) Structurally, it consists of only three segments. The head is well armed with two rows of hooklets, thirty to forty in number, seated on a rather swollen rostellum. Behind the hooklets are four suckers, radially disposed. The neck, which is not segmented, connects the head with the first proglottis. This proglottis does not contain sexual organs, but is succeeded by a second proglottis which is more highly developed, and contains ova, oviduct, and a germinal layer.
- When ripe, the terminal proglottis breaks off, and the ova are liberated into the larger intestine and extruded with the faeces. These may be discharged on to green vegetables, or they may contaminate drinking water, and thereby be directly ingested by man; but more commonly they are ingested by sheep or cattle.

In Australia, where surface waters are largely used for domestic purposes, the ova, after being scattered on the soil, are washed into the water supply, and are a constant danger to man.

The intervention of animals so closely associated

with the domestic and economic life of man, is an essential factor in the genesis of this serious human infection.

So far as we know, the only animals in which the *Taenia Echinococcus* has been found, are the domestic dog, the wolf, the jackal and the fox, On the other hand, the bladder stage is more widely distributed, having been found in monkey, sheep, ox, pig and in cat. Of the domestic animals, the sheep, ox and pig are the principal hosts of the larval stage, and in this country the sheep is the one of most significance.

Braillford (3) first called attention to the possibility of the fox being a carrier of the infection in this country. He examined the intestines of a number of foxes, but was not successful in finding the parasite.

Town dogs appear to be relatively free of the infestation. Braillford did not find the *taenia* in any one of the hundred dogs he examined in Birmingham in 1920, although 65% of them were harbouring other tape worms.

These statistics suggest that, either the rules governing slaughter houses are more strictly enforced in towns than in rural districts, or else that slaughter houses are not such a frequent cause of dissemination of the disease as some authorities would have us believe.

Krabbe discovered the parasite in 25% of dogs in Iceland, whereas the percentage of infested dogs in Australia varies, in different districts from 15% to 100%.

Examination of the carcasses of sheep killed in the Keswick slaughter houses over a period of six months failed to find a single instance of Hydatid disease.

Geographical distribution of the disease.

It will be seen from the foregoing remarks, that there are certain essential factors governing the existence of the parasite, and on these factors will depend the geographical distribution of hydatid disease.

(1) Sufficiency of dogs infected with *Taenia Echinococcus*, by which means the supply of ova is ensured.

(2) An abundance of animals, such as the domestic herbivora, particularly sheep, capable of serving as intermediate hosts.

(3) Conditions favouring the entrance of *Taenia* ova, into the alimentary canal, either of man, or of the intermediate host, by which the supply of *Taenia Echinococcus* is kept up.

Statistics indicate that Hydatid disease is most prevalent where these conditions are fulfilled, e.g. Iceland, Australia and New Zealand. There are no reliable statistics for Great Britain available, as the disease is not notifiable, but we do know that in Orkney and the Shetlands, Hydatid disease is endemic, and that in these places the essential governing conditions exist.

(1) As regards the sufficiency of dogs.

In England there is one dog to every 50 inhabitants, whereas in Iceland the proportion is 1.5. In Australia the proportion is about 1.3.

(2) The proportion of sheep to man, is far higher in Australia than elsewhere. In 1893 there were 3000 sheep and 300 cattle to every man, and it has been estimated that 22 to 30% of these were infested.

(3) As regards the access of ova to the human alimentary canal. The close relationship of dog to master, found in all people who lead such a lonely life as a shepherd, needs no comment. In this country also, dogs in farm houses often feed from the same platter as the family. They are allowed to lick the children's hands and faces, to be nursed and petted, and often enough, to share the same bed as their master. In this respect also, it seems possible that cats may be a not uncommon mode of human infection. Deve has infected the cat experimentally, and cats are frequently fed on raw sheep's lungs.

The Hydatid Cyst.

As has been mentioned, the egg gains access to the human body through contaminated food or water, or by direct contageon with an infected dog.

When the egg comes in contact with the gastric juices, the protecting shell dissolves, the embryo escapes, and by means of its hooklets and succers, gains anchorage to the mucous membrane of the stomach or small intestine.

No one has yet been able to trace the passage of the embryo through the gastric wall, but it was traced by Leukart in the case of *Taenia Soleum*, and it may be presumed that the same process is followed by the *Taenia Echinococcus*, and access gained to the venous or lymphatic channels. It seems probable however, that lymphatic invasion is extremely rare, as I can find no trace of any case occurring in lymphatic glands, other than one recorded by Harrara Vagas. (4).

The embryo after entering the portal system, is carried to the liver, and in this organ 75% of hydatid manifestations occur. (9)

The embryo, however, which at this stage is only three times the size of a red blood corpuscle, may pass up to the right heart and thence to the lungs, where 8% of cases manifest themselves.

The remaining 17% of cases are carried from

the lungs back to the left heart and thence to the systemic circulation, and cause cysts in almost any part of the body. The commonest sites however are, the muscles 5%, spleen 3%, kidney, 2%, brain 1.5% and bone 1%.

Growth of the resting embryo.

After coming to rest in one or other organ, the embryo starts to increase in size. The external envelope becomes laminated, and the contents, owing to partial liquifaction, become transparent. Fluid accumulates within the bladder, and instead of a solid spherical body, the organism becomes a vesicle containing an ever increasing quantity of poisonous fluid. An internal lining then appears, constituting the germinal layer, called by Huxley and Boyd the "endocyst." From this layer are budded off new heads or scolices. Each scolex develops into a little cup shaped process called by "Boyd" (5) the "brood capsule." Each capsule may contain many scolices. In addition to forming new heads, the endocyst may give rise to numerous daughter cysts which usually develop within the main cyst, but sometimes outside it. New scolices develop in comparatively few of these daughter cysts, the majority of them remaining sterile.

Further extension of the cyst takes place by a quiet pressure necrosis of surrounding tissue, and its replacement, *pari passu*, with fibrous tissue. This fibrous tissue coat, developed from the organ in which the cyst is growing, and not in any way an organic part of the parasite, is known as the ectocyst. Nevertheless it is of importance, as it is the means of nutrition of the true cyst.

Macroscopically, it at first resembles coagulated white of egg, but as it increases in age, it becomes dense and leathery, and acquires the consistency of a tumour.

Age incidence.

The growth of the cysts in the human body is extremely slow, and their latency is characteristic of the disease.

It has been stated by Dew, (6) that the majority of hydatid infestations, like those of other parasites, take place in childhood, so that the cyst is often nearly as old as its host. This contradicts the general opinion, expressed by Vagas, that 50% of cases are fatal within five years of infection, and suggests that the absence of symptoms caused by the latency of the cyst, prevents the early diagnosis of the disease. It is in fact, the complications of hydatid disease, that most often leads to the discovery of the presence of the cyst.

The cyst may exert pressure upon any structure, and since almost any organ may be involved, it can be understood that bizarre and protean manifestations may be produced. Large cysts can be tolerated, and this is not surprising when one assumes that many infestations occur during the most rapid growing period of life, and the extremely slow growth of the cyst allows efficient compensatory changes in the organ concerned; so much so in fact, that the cysts are shut off from the body fluids of the host by a relatively impermeable fibrous adventita.

Ultimately however, some complication is bound to occur, and the relative frequency of such complications increases with the age of the host; and in fact are rare before the age of 25.

Practically all complications depend on the escape of fluid from the cyst, the toxicity of which will be referred to later.

Hydatid Disease in Keswick.

The three cases of hydatid disease to which I am now going to refer, occurred in Keswick during the last seven years, and demonstrate the points already mentioned.

They are of interest, as there is a familiar and also a geographical relationship between them. All cases were in persons occupied in the sheep farming industry, although cases (2) and (3), had retired from farm life for eight years.

Case (1)

Miss L. H. Age 21. Farmer's daughter, living in an isolated area, under somewhat primitive sanitary conditions. The water supply was from a spring in the mountain side on which sheep were kept. Four or five dogs were constantly employed on the farm, and the girl had been brought up in close domestic contact with the dogs.

In January 1922, when thirteen years old, she was first seized with attacks of severe dyspnoea, followed by physical signs of a large pleural effusion on the right side. Two pints of clear fluid were aspirated, and the specimen found to be colourless, faintly alkaline, and containing neither albumin or cellular deposit. It gave a reaction to Fehling's solution, to about the same extent as that of normal cerebro spinal fluid. Neither scolices or hooklets were found.

During the next four months re-accumulations of fluid took place, and as a result of frequent aspirations, the fluid became turbid, and necessitated drainage and rib resection. At the operation a large cavity was found in the chest but nothing but semi-turbid fluid escaped.

The wound took six months to heal, and during this time she was the subject of sudden severe attacks of dyspnoea, accompanied by extreme cyanosis.

For the next two years she enjoyed fair health, and a gradual return to strength. The physical signs in the chest were as follows:- Diminished movement, with a chest measurement $1\frac{1}{2}$ " less on the right side than on the left. Percussion dullness up to the fifth rib. Broncophony at the right base, and Skodaic resonance at the right apices. The apex beat was $3\frac{3}{4}$ " from the mid line. There was no general emaciation, no pyrexia, and no night sweats. Sputum examination for T.B. was negative. No liver, spleen, or glandular enlargement.

In March 1925, she again had attacks of severe dyspnoea, with pain over the base of the right lung. The right chest now measured three inches greater than the left, and there was bulging and prominence of the right breast. Clear fluid was again aspirated, and symptoms subsided

till the next year, when the old scar broke down, and a little pus and four small cysts, the size of peas, were extruded.

During the next six months she had frequent attacks of pain and dyspnoea, and coughed up numerous daughter cysts varying in size from that of a pea to a cherry. These daughter cysts contained a little clear fluid. Most of them were sterile, but scolices were found in a few.

Thereafter she made a wonderful recovery, putting on weight and being entirely free of symptoms. The eosinophyl count at this time had fallen from 12% to 8%.

In July 1929, a series of severe attacks were followed by bulging and breaking down of the wound. The wound was freely re-opened and a cavity the size of an orange was found, containing free fluid but no daughter cysts.

This cavity did not communicate with a bronchus, and the symptoms did not clear, and she continued to expectorate cysts.

In August 1929, she was admitted to the Edinburgh Royal Infirmary, and after X-ray examination, the chest was explored by Professor Fraser.

A portion of the seventh rib was removed and the cavity entered. Several small daughter cysts were found, and the wall of the cyst, which had the thickness and density

of liver, was stripped away. The adjacent lung was found to be collapsed and necrotic, and was also so dense that further exploration of the lung was impossible. The operation was followed two days later by several attacks simulating collapse, and not accounted for by the shock of operation.

She made a fairly good recovery, however, and was able to return home in October 1929.

Her improvement thereafter however, was of short duration, and she continued to expectorate cysts, and to have sudden attacks of dyspnoea and collapse. These attacks were characterised by an increase in the pulse rate, flushings and sweats alternating with cyanosis and pallor, also pruritis and erythema of the skin. The pulse at times was almost imperceptible, and eventually she died during one of these attacks in June 1930. The cause of death was attributed to Hydatid anaphylaxis.

This case leads to the discussion of two important aspects of hydatid disease viz: (1) Possible complications and sequelae of the hydatid cyst. (2) Hydatid anaphylaxis, and the properties of hydatid fluid. It also raises all the difficulties of diagnosis, and is an example of how a case of a cyst in the pleural cavity should not be treated in the early stages, were it correctly diagnosed at that time.

Complications.

Pleural and pneumonic infestation are not uncommon, and is caused in one of two ways. Either the primary cyst is in the liver, and by its growth eats through the diaphragm and finally penetrates into the pleural cavity, or else the fertile simple cyst ruptures into a vein and enters the circulatory system, the so-called "metastatic secondary echinococcosis" (Dew) (7).

In this case also the most common organ infected will be the lung, as the scolices will pass through the right heart and enter the pulmonary artery and so reach the lung.

The first method is the most common, and the cyst after penetrating the diaphragm and entering the pleural cavity, will push back the neighbouring structures, and surround

itself with its pericystic membrane. The lung tissue suffers from this pressure and atrophies.

The bronchi are flattened and finally rupture and open into the "hepato bronchial fistula."

The contents of the cyst can thus be evacuated and result in a natural cure. This evacuation will very likely be stained with bile. In the case just mentioned, bile was not found in the expectoration till two years after the onset of symptoms. This might suggest that the penetration of the diaphragm occurred from above, and that it was a true case of secondary echinococcosis. At no time was any liver enlargement detected.

Symptoms of pulmonary cysts usually start about two years after implantation of the cyst, and are characterised by pain, cough and haemoptysis. Australian statistics give an 8% mortality in adults, and a 5% in children.

Rupture into a bronchus is the natural end to most pulmonary cysts, because, by the time the cyst reaches a size of 4 or 5", it nearly always comes in contact with a large bronchus, the erosion of which causes a small part of the cyst wall to become unsupported. Following a cough or muscular effort, the cyst wall gives way, and a hepato-bronchial rupture results.

When this accident occurs, the escape of fluid may be of so great a quantity as even to drown the patient. There

is also the possibility of the impaction of one or more cysts in the trachea, and asphyxia resulting.

Miss L. H. was the victim of this distressing condition on frequent occasions, and the physician was almost powerless to help her.

As a rule however, the severity of the symptoms is out of proportion to the amount of fluid expectorated, and it is probable that the persistent frothy sputum, dyspnoea, cyanosis, and general collapse, so often seen in the above case, was largely attributable to anaphylaxis.

The chances of natural cure resulting from rupture into a bronchus, largely depend on the situation of the cyst as regards the possibilities of drainage, and also on the size of the cyst and the thickness of the adventita, on which depends the possibility of complete collapse of the cyst and disintegration and expectoration of the cyst wall.

The element of bacterial infection after the rupture will of course, also effect the chances of recovery. At first in a simple rupture, the chances of infection will depend on the condition of the patient, and presence or absence of a super-added bronchitis. Sooner or later however, infection is bound to occur, and a pyo-chylo-thorax will result. Here again recovery will largely depend on the

position of the cyst as regards drainage.

In the early stages of Miss L.H.'s illness, prior to the discovery of the correct diagnosis, aspiration was employed for diagnostic and therapeutic means; and it would be well to review the dangers of this operation in this condition.

Dangers attending aspiration.

One must realise that the contents of the cyst are under pressure, and that rupture is always imminent. Under these conditions the mere pressure of the needle is very likely to hasten this catastrophe, and cause a large tear in the cyst wall like that produced by pricking a toy balloon, and thereby incur the possibility of the drowning or asphyxia of the patient, as well as the probability of causing severe collapse by the sudden release and absorption of the poisonous hydatid fluid.

Another danger of aspiration is the possibility of leakage of the cyst contents into the surrounding tissues, through the channel made by the aspirating needle, and thereby causing dissemination of the disease.

These dangers are so fully recognised by the surgeons of Australia, that they recommend that no doctor attempt aspiration, until he has resected a rib, and is prepared to make an incision into the sac, should it prove necessary.

Hydatid fluid. As has been mentioned in the case described, the specimens of fluid aspirated, puzzled the pathologists. Chemically it appears to be fairly harmless; it is clear in appearance, faintly alkaline in reaction, and contained a small quantity of sugar and chloride. Herein it may be compared to cerebro spinal fluid and also to amniotic fluid. Like amniotic fluid also it undoubtedly has a nutritive value.

In spite of its innocuous appearance however, it is far from harmless, and when injected into animals, it has been shewn to act as a cardiac poison, producing a diastolic stand still, a lowering of the blood pressure and temperature, and other symptoms of depression of the vital functions. (12)

Dessy and Marottas, experimenting in the Argentine, found that if a previous injection of adrenalin was made, the symptoms did not occur, and it seems reasonable to suppose that the distress caused by the poison is largely due to the fall in blood pressure.

A patient suffering from hydatid disease may absorb varying amounts of hydatid fluid. This absorption will commence at a very early stage, during the development of the cyst and before the impermeable laminated membrane is fully developed. As a result of this absorption,

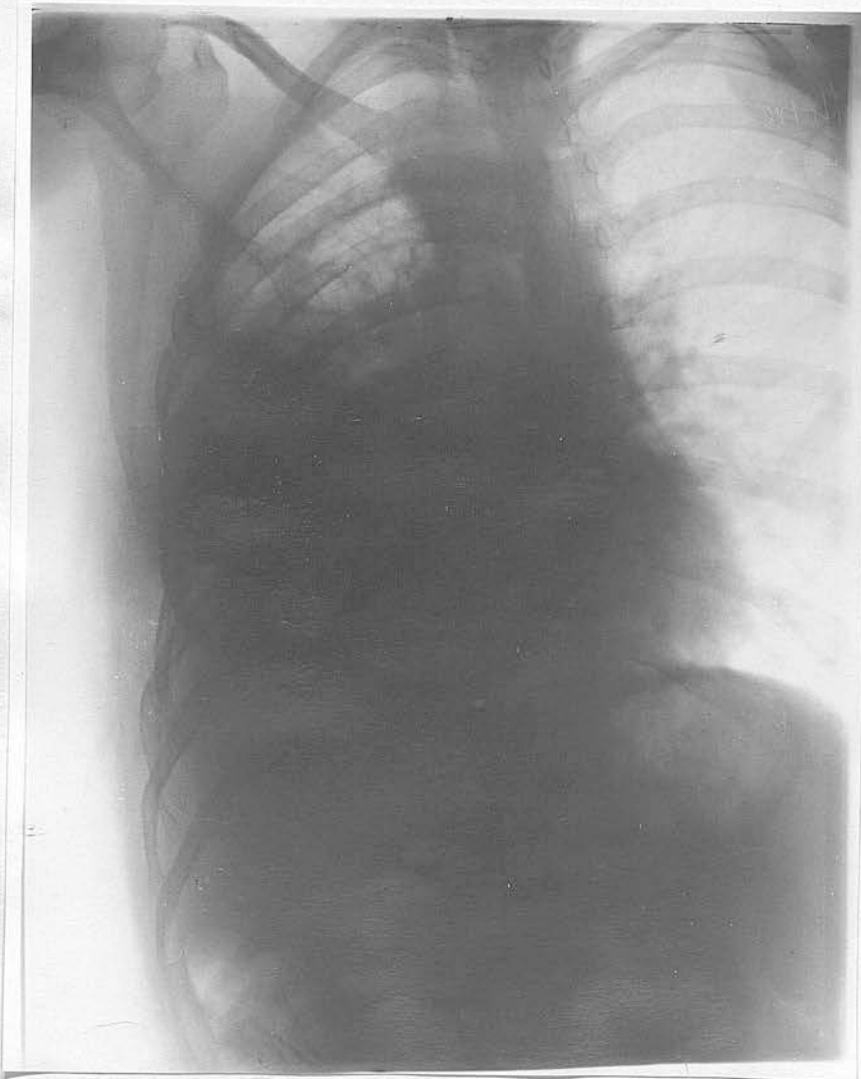
specific sensitisation of the cells of the host takes place, and if at any later period even small amounts of hydated fluid are released into the system, anaphylactic symptoms will be produced.

The degree of sensitisation varies greatly, and depends on the doses of poison absorbed, and the intervals between these doses. There is probably some absorption from even simple cysts, where there is no evidence of leakage.

Clinically, the symptoms may be either mild or severe. In the former case there will probably be cutaneous manifestations in the nature of an erythema, and also attacks of faintness, pallor, dyspnoea, vomiting and irregular pyrexia. The severe cases are characterised by extreme collapse, small pulse, severe dyspnoea, cyanosis, oedema, syncope, and a rapid fall in blood pressure. As a general rule an injection of adrenalin works like a charm, but in Miss L. H. it did not alleviate the symptoms, and did not even raise the systolic blood pressure.

The cause of the severe attacks are usually attributable to the rupture of a cyst into the surrounding tissues or into a bronchus or bile duct, or as has already been mentioned, the same condition may be produced by an exploratory puncture.

Deve, in 1911 pointed out that general anaesthetics abolish anaphylaxis, and it was observed that Miss H. did not suffer from shock till two days after the operation: this was almost certainly a case of delayed anaphylaxis.



Secondary echinococcosis.

This nomenclature was first used by Dew in Australia, and Deve in Europe, to denote the multiple dissemination of the cysts, as a result of rupture of a primary cyst into the blood stream. It might also be applied to the rupture of a liver cyst into the general peritoneal cavity, and dissemination of the disease over the abdomen. This phenomenon is the most likely origin of the not uncommon pelvic hydatids, and was the explanation put forward by John Hunter in 1793.

9 Rupture of the cyst, or aspiration by trocar^h does not necessarily cause the death of the parasite. The parasitic elements have enormous powers of resistance, and can survive, and ultimately develop into new cysts, often at a distance from the original site. This persistence is demonstrated by Deve's (8) experiment, in which he produced cysts in a rabbit by injecting washed living scolices under the skin.

Hydatid disease of the liver.

Statistics shew that 75% of hydatid disease involves the liver: 9% the lung, 5% muscle, and 4% the abdominal viscera. About 1% are found in the pelvis. (9)

The right lobe of the liver is the more commonly affected, because the right portal vein runs a less tortuous course than the left, and is also a larger vessel and drains a larger area of the stomach and intestines.

As a general rule the cyst is a solitary one, though sometimes multiple, and occasionally the liver is infiltrated with numerous hydatids.

Unlike the lung, the liver has plenty of room for expansion, and hydatid disease in this organ is accompanied by a general hyperplasia of the organ, and symptoms of liver deficiency are thereby prevented.

The possible complications of a liver cyst will depend on its situation within the organ. Most commonly it grows down from the abdominal surface, and may rupture into the peritoneal cavity. Frequently it grows upwards, penetrating the diaphragm, and becoming a hepato-pulmonary cyst. It may extend backwards into the lumbar region, or it may be central.

This last situation seems to be the most favourable to degenerative changes, and post mortem

examination has not infrequently revealed old cysts, the existence of which has been unsuspected during life, and which have undergone sclerosis or even calcification due to the deposit of lime salts.

The symptoms of an hepatic cyst are illustrated in the following case:-

Case 2. John A. Age 22. Chemist. April 1929.

He lived on a farm until he was 18 years old, and has since lived in Keswick. His mother was operated on five years ago for the removal of a hydatid cyst of the broad ligament. Miss L. H. is a distant relation, and a frequent visitor when he lived on the farm.

He complained of lassitude for some months and a feeling of fullness in the stomach. On examination there was an obvious fullness in the epigastrium, slightly to the right of the mid line. There appeared to be a raising up of the lower right ribs. A tumour was felt on palpation, and appeared to be growing down from under the costal margin. It was smoothe in outline, and dull on percussion, and was only slightly tender. A definite "hydatid fremitus" was elicited, compared by Davaine to the quiver produced by percussing a spring mattress. The dermal reaction to hydatid fluid was positive.

An operation was performed, and a cyst the size

of an orange was enucleated. The cyst wall was found to be composed of fibrosed liver tissue, and contained the typical clear fluid, and five daughter cysts. Hooklets and scolices were not found. There was no evidence of the existence of a second cyst.

He made an uninterrupted recovery, but the dermal reaction is still positive.

Pelvic Cysts.

The third case of Hydatid disease is also of interest. Mrs A. age 54, the mother of John A. and likewise a frequent companion of Miss L. H.

In 1924, three years after she had left her farm, and come to live in Keswick, she complained of constant discomfort, and a feeling of fullness in the lower abdomen. She otherwise felt fairly well, and there were no symptoms suggesting hydatid anaphylaxis. On examination a pelvic cyst was found, and presumed to be of ovarian origin. On operation however, it was found to be growing in the broad ligament on the right side. The cyst was removed and found to be a hydatid, containing twenty or thirty daughter cysts.

The rest of the abdomen was examined and found to be healthy. There was no liver enlargement, and no evidence of an old or degenerated hepatic cyst. There were no adhesions round the cyst. She made a complete recovery, and still remains well. Her eosinophyl count after the operation was 2%, and it has not altered since. Her dermal reaction is still positive.

In 1929, five years later, she had symptoms of gall stones, but these quickly disappeared.

Pelvic cysts are somewhat uncommon, only one in

1000 occurring in this region. They are more common in the female than in the male in the proportion of 1.3. The pelvic cellular tissue is the most commonly affected, and the ovary the rarest.

Their origin may be due either to rupture of an hepatic or splenic cyst, or to a metastatic blood carried implantation. If this case were due to blood carried means, one would expect to find cysts in other organs particularly in the lungs. If on the other hand the infection were secondary to a ruptured hepatic cyst one would expect to find suggestive facts in the past history of anaphylactic symptoms at the time of the rupture of the primary cyst, and also one would expect to have found some sign in the liver or spleen at the operation.

Of the two alternatives, the second is the more probable, and the patient did not suffer sufficient reaction for the rupture to have made any impression on her.

Owing to the rapid and complete recovery from the initial rupture in most instances, it is not uncommon for the symptoms to be misinterpreted, and the significance is not recognised until a later period.

Very often however the rupture of an hepatic cyst into the peritoneal cavity, is attended by serious

complications.

First of all of course, there is the danger of the absorption of a large quantity of hydatid fluid. The next danger is that the sudden release of the pressure of the hydatid within the liver, may be attended by a rupture of one or more small bile ducts, and the consequent escape of bile into the abdominal cavity.

At first the peritoneum is extremely tolerant, and a biliary ascites develops slowly over many months. As a rule however the collection becomes localised by adhesions. Sooner or later infection is almost certain to occur, and a chole-peritoneum becomes a chole-peritonitis, with the correspondingly grave symptoms.

Dew (6) states that secondary echinococcal cysts are always multiple, but this case tends to contradict this statement.

He also states that secondary cysts are of extremely slow growth, and that there is always a latent period of five to twelve years before they produce symptoms.

It would appear then, that the pelvic cyst started growth before 1918, and her primary cyst was even older. It is probable that the son was infected from the same source as the mother, and his cyst must therefore

have been at least eleven years old before it gave rise to any symptoms.

Miss L. H's cyst ruptured into the pleura in 1922, so that it is probable that her primary liver cyst (if she had one) was implanted also before 1918, and all three cases received infection from the same source.

We were not able to trace the infection in any of the farm dogs, but our examination was probably too late, and the dog that caused the trouble is long since dead.

Our examination of the slaughter houses has also been negative, and no echinococcal infestation has been found in any sheep, though the "liver fluke" was not uncommon.

Diagnosis.

The three cases just reported illustrate many of the difficulties with which we meet, in attempting to diagnose hydatid disease. In only one of these cases was the true diagnosis made before operation.

I believe that many general practitioners regard hydatid disease as an extreme rarity in this country, and most of us are quite ignorant of the auxilliary methods of diagnosis.

In Australia where the condition is so much more common, it is very likely that the pulmonary cyst would have been readily diagnosed, whereas it baffled four general practitioners ~~in this country~~ and three specialists for a period of four years.

Of the auxilliary methods, the most useful and the most easily performed, is the dermal test introduced by Casoni. It is also the most reliable. It consists of the intra-dermal injection of .5 c.c. of bovine hydatid fluid, which has been filtered, and to which has been added one drop of phenol to 20 c.c. of fluid. If the test is positive, a patch of rusty reddish oval spots will occur at the sight of the injection. This is accompanied by local œdema and a local rise in

temperature. This test was first suggested to Casoni by the rash accompanying hydatid anaphylaxis, and has been elaborated by Gasbarrini, Bull and others. Dew states that in uncomplicated cysts ^{there is} an immediate reaction and a delayed phase in 90 to 95 per cent of cases, whereas with the presence of ruptured cysts the ^{delayed} phase may be absent.

In no one of the three cases reported was any delayed phase observed, and all three gave definitely positive results. It is interesting to note that the test is still positive in the liver and pelvic cases.

The occurrence of three cases in two families naturally caused some alarm amongst the relatives, and by using the dermal test, one was able by the negative results obtained, to reassure them.

The compliment fixation test introduced by Bordet and Genou, (10) is of less value, as it is frequently negative in cases of unruptured or dead cysts. It depends on the fact that the same compliment will unite with both haemolytic and bacteriolytic immune bodies, and is used to detect the presence of immune bodies in the serum of hydatid infected patients.

This compliment fixation test was positive in the case of Miss L.H., but was not tried in the other two cases.

The percentage of eosinophyls in the blood is also useful as a confirmatory factor, as the majority of cases show a marked increase. This however is not pathognemonic of hydatid disease, as a similar increase is to be found in most helminth infections as well as in certain skin conditions, e.g., psoriasis, in lymphadenoma, leukaemias, and in asthma. In one case of bronchial asthma, I found an eosinophyl count of 42%, and in another a count of 25%.

The highest counts found in the hydatid cases were

12%, 14% and 2%.

Thoracic cysts are most easily detected by X-rays, particularly before rupture. Such a patient may be sent for radiographic examination on account of hæmoptysis where tubercle is suspected, or perhaps physical examination has suggested the possible presence of a neoplasm.

The simple cyst will shew as a uniformly dense ovoid shadow, with a well defined outline, and with the surrounding lung apparently healthy. These characteristics distinguish it from the neoplasms, aneurisms, interlobar empyemata and pyæmic abscesses. Great difficulty

however, may be found in determining the nature of a shadow case by a cyst which has ruptured. The cyst may still have retained a portion of its contents, and a fluid line be shewn, and this may be mistaken for a pleural ring. A ruptured cyst of some duration is attended by considerable fibrosis of the surrounding lung, and an accurate diagnosis by skiagram alone, will then be impossible.

In any case however, the photograph will be enough to raise suspicions and indicate the employment of one or other of laboratory tests.

It will be seen from the foregoing remarks, that the diagnosis of hydatid disease presents many difficulties, especially in countries where the disease is comparatively uncommon, and the majority of medical men are not familiar with the condition.

Hydatid disease may occur in any organ of the body, and may thereby simulate almost any other pathological condition.

A thoracic cyst may displace the heart, the trachea or the aorta. It may rupture into the pericardium,

the trachea or into the pleura. The abdominal cyst gives the identical physical signs of any other abdominal cyst, and may grow as large as any ovarian cyst. Deve reported a case of an abdominal hydatid that contained 22 pints of fluid.

On account of the size of the carotid arteries, the brain is the most common site of secondary metastatic blood borne echinococcal cysts, and a cyst in this organ will give the identical pressure symptoms of a neoplasm.

It is not then surprising, that except for hepatic cysts, where the differential diagnosis is more limited, that so many hydatids are only correctly diagnosed by accident, either at radiographic examination, or at an exploratory laparotomy.

Treatment.

The treatment is essentially surgical, and enucleation of the cyst the ideal result. The possibilities of this depend of course, on the situation and the organ infected.

Deep X-ray therapy proved disappointing, as also did radium. (13)

Attempts by Dufour, to produce an hydatid antitoxin were unsuccessful.

Summary.

These three cases present a number of rather unusual features, and also impress on one's mind the seriousness of the disease.

Two of the cases shewed symptoms at the early ages of 13 and 22 years. It is an undisputed fact that infection in a very large proportion of cases takes place in childhood, but it is very rare for a host to shew signs of infestation before the age of 25.

Another interesting feature of this series of cases is, that although there is no record of hydatid disease in the Keswick area during the past 35 years, three cases occurred between 1922 and 1928, and all these three cases were related and very likely infected from the same source.

When one considers the possibilities of a mass infection, it is difficult to understand why serious epidemics do not occur.

The great danger of hydatid disease lies in the complications caused by rupture, and the most important duty of the physician is the correct diagnosis, wherever possible, before the occurrence of this catastrophe.

I believe that in this respect, Casoni's dermal test should be more widely employed. The incidence of

one case in a family or farmstead, should be followed by the use of the skin test on each remaining member of the family.

The question may arise as to whether a man who has been apparently successfully operated upon for the removal of a hydatid cyst, is acceptable as a first class life for insurance purposes, and how long after operation one would expect to find a positive skin reaction?

In the case of the liver cyst, the test is still positive two years after operation. We are justified, I think, in believing that the man is cured, and that the positive test depends merely on the presence of the alien protein in the blood serum.

In the case of the pelvic cyst however, the situation is a very different one. It is almost certain that her original primary cyst was in the liver or spleen, and we cannot be sure that this cyst is not still alive. It is eight years since her operation, and one would have expected after this time, at least some delay in the appearance of the wheal in Casoni's test.

There was no such delay, and she has also during the last three years, been the subject of two attacks simulating biliary colic. We cannot be sure

that these attacks may not be of hydatid origin.

Casoni's test is not therefore of any prognostic value.

Conclusions.

- (1) Hydatid disease is not so rare as is generally believed, and is far more common in England than one realizes. The disease is not notifiable in England, and it is not therefore possible to obtain statistics. Nevertheless it is responsible for four hundred deaths a year in Great Britain, and post mortem examinations reveal the presence of hydatids in 1.5% (Murchison).
- (2) The fact that eight physicians, seven of them of wide experience, failed in the diagnosis of a pulmonary cyst, even with the report on the examination of the fluid available, suggests that the teaching of the features of hydatid disease, does not receive its proper share in our medical schools.
- (3) Greater efforts should be made to eradicate the disease by:-
 - (1) Compulsory notification.
 - (2) Education of the public as to the danger of too close proximity of children to domestic animals.
 - (3) Examination of the water systems of country farms, where reservoir supply is not possible.

- (4) Careful inspection of slaughter houses and farms in areas where cases are notified, with a thorough investigation by the sanitary authorities into all such cases.
- (5) The teaching of butchers to recognise the hydatid cyst, and the notification of any infected carcase.
- (6) Forbidding the use of the entrails of cattle as a ground manure.

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